Part (1): ANESTHESIA

PRE-OPERATIVE EVALUATION & PREMEDICATIONS (***)

All physicians involved in the care of patients - internists referring the patients, surgeons operating on them, and anesthesiologists providing anesthesia and life support during the intraoperative and postoperative period - share the common goals of producing the best possible surgical outcome and minimizing risks to the patient's survival and well-being.

Evidence indicates that perioperative management affects the outcome for better or worse, and central to achieving the best possible outcome is a thorough preoperative evaluation intended to:

Goals

a) *Identify the health problems that place the patient at risk.*
b) *Resolve or control the disease as well as possible.*
c) *Define a management plan that minimizes pre-, intra- and postoperative risks.*
d) *Preoperative visit to provide reassurance to the patient.*
e) *To prescribe premedication drugs.*

*** A Complete Preoperative Evaluation Consists of:**

- *A systems-oriented history and physical examination:*

1- History of previous adverse effect to anesthesia: prolonged apnea or paralysis, delayed recovery, nausea& vomiting, jaundice, postspinal headache... ect.
2- Drug and allergy history.
3- Current drug therapy or abuse.
4- History of organs dysfunction or systemic diseases: CNS, CVS, respiratory, liver, renal, endocrinal, coagulation, reproduction, dentition and musculosceletal systems.
5- General examination: CNS, CVS, lungs.
6- Examination of the upper airway and/or the back (in spinal anesthesia).
7- Any necessary and diagnostic tests: it includes general routine investigations (Hb, blood chemistry, coagulation study, urinalysis, ECG, chest x-ray).
8- Specific investigations according to the patient's condition (liver, renal or pulmonary function tests and others).
9- Then, formulation of an anesthetic plan obtained from the patient.

*** Pre-Operative Medications:**

It includes psychological and pharmacologic components. The drugs used in the preoperative visit are used with one or more of the following goals:

1- Sedation
2- Anxiolysis
3- Analgesia
4- Amnesia
5- Antisialogogue (drying of secretions)
6- Prevention of allergic phenomena.
7- Reduction of vagal activity
8- Decrease gastric fluid volume.
9- Increase gastric pH
10- Attenuate stress response
11- Prevent nausea and vomiting
12- Decrease anesthetic requirements
13- Facilitation of anesthetic induction.
14- Antihistamines.

* Caution should be exercised when administering premedicants to certain patients:

- *Pediatric patients under 1 year of age.*
- *Geriatric patients.*
- *Patients with intracranial pathology*
- *Critically ill or hypovolemic patients.*
- *Cardiac patients: with dysrhythmias or myocardial infarction.*

*** Commonly used preanesthetic drugs:**

1- Benzodiazepines:

- They are useful.for anxiolysis, sedation and amnesia.
- They have a high therapeutic index with minimal cardiorespiratory depression.
- Disadvantages: excessive and prolonged sedation, pain on injection.

a) Diazepam (Valium):

- Effective premedicant with good sedative, anxiolytic and amnesic properties.
- Better given orally as parenteral injection may cause phlebitis and pain.

b) Lorazepam (Ativan):

- 5-10 x more potent than diazepam
- Produces heavy sedation and reliable amnesia
- Has long duration of action (half life 10-20 hr.)
- Does not produce phlebitis or pain on injection.

c) Midazolam (Dormicum):

- Has 3-4 times the potency of daizepam.
- Water soluble, rapidly metabolized with half life of 1-4 hr.
- Displays very good sedative and amnesic properties.

2- Barbiturates:

- Secobarbital and pentobarbital are occasionally used.
- They provide reliable, and safe sedation, but are not as specifically anxiolytic as benzodaizepines.
- Effective when administered orally.
- Disadvantages: lack of analgesia, disorientation, absence of specific antagonist, stimulation of hepatic microsomal enzymes.
- Contraindicated in porphyria → acute exacerbation.

3- Opioids:

- They are used for premedications when analgesia is needed e.g. a patient with a painful condition or one who will require potentially painful monitoring lines or regional anesthetic techniques.
- Absence of direct myocardial depression.
- Can be administered through different routes: oral, IM, IV, spinal rectal, transnasal, transdermal, transmucosal and lolly pop (for pediatrics).

*** Side Effects of Opioids:**

- Respiratory depression
- Nausea and vomiting
- Potential for choledochoduodenal sphincter spasm
- Urinary retention
- Excessive sedation
- Orthostatic hypotension
- Pruritus
- Bradycardia
- Constipation
- Delayed recovery

a) Morphine:

- Best given preoperatively i.m (0.1 mg/kg)
- Peak effect in 30-45 min. and duration of action is about 4 hr.
- Can be given by other routes iv., orally or suppository.
- Caution is advised in the elderly, and O2 supplementation is advised.

b) Meperidine:

- Given preoperatively intramuscular 1 mg/kg
- Peak effect in 1 hour, duration of action 2-4 hr.
- Similar in structure to atropine thereby may cause tachycardia.

4- Anticholinergics:

- Most commonly used preoperatively to dry oral secretions, prevent vagally mediated bradycardia especially in pediatrics and production of sedative & amnesic effect.
- They are not effective in ↑ gastric pH or ↓ gastric volume.
- **Disadvantages:** CNS toxicity, tachycardia, mydriasis & cycloplegia, ↑ body T°, drying of airway secretions, relaxation of lower esophageal sphincter and ↑ physiologic dead space.

Comparative Effects of the Anticholinergics

	Atropine	Scopolamine	Glycopyrolate
-Antisialagogue	+	+++	++
-Sedative,amnesia	+	+++	0
- ↑ gastric pH	0	0	±
-CNS toxicity	+	++	0
-Mydriasis	+	+++	0

5- Prophylactics against aspiration pneumonitis:

- Many patients presenting for operation may be at increased risk for regurgitation and aspiration of gastric contents: e.g. parturient, obese, patients with difficult airway& gastroesophageal reflux.
- The risk of adverse pulmonary sequelae is greater when:
  # Gastric volume is greater than 25 ml (0.4ml/kg).
  # pH is less than 2.5
- The patient should be fasting for 6-8 hr.

a) Cimetidine:

- An H2 receptor antagonist
- Given the night before and the morning of surgery to produce a reliable increase in gastric pH.

b) Ranitidine:

- Has a lower incidence of side effects than cimetidine.

c) Famotidine:

- A newer H2 receptor antagonist. - Duration of action is 10 - 12 hr.

d) Metoclopramide:

- A dopaminergic antagonist that stimulates gastric motility speeding gastric emptying.
- It increases the tone of the lower esophageal sphincter
- It has no effect on gastric fluid pH, an H2 antagonist should be used
- Its side effects include exrtapyramidal syndrome.

e) Non-particulate antacids:

- Like sodium citrate which is effective in immediately increasing gastric pH.

6- Others:

- The patients' regular medication: It is a common practice to continue all regular medication throughout the perioperative period. The patients usual medications may be given along with the other preoperative medications with a few sips of water.

a) B-blockers:

- Rebound hypertension or ischemia may occur if stopped abruptly.
- Intraoperative bradycardia may be treated with atropine.

b) Calcium channel blockers:

- Myocardial ischemia may occur with abrupt cessation

GENERAL ANAESTHESIA

Balanced Anaesthesia

It means avoiding the need for deeper levels of anaesthesia, by using muscle relaxants and powerful analgesics in modern G.A. to provide the ideal conditions to perform a safe surgical procedure. This includes hypnosis, analgesia, areflexia and muscle relaxation.

All these conditions can be achieved by the use of one drug as ether or thiopentone, but will require the use of a massive dose to achieve adequate depth of anaesthesia, which may become harmful or toxic. However, with the use of specific drugs to produce specific effects to provide the desired surgical conditions, deep anaesthesia can be avoided, e.g. by using:

1. strong opioids for analgesia
2. muscle relaxants for relaxation and areflexia
3. minimal inhalational or I.V anaesthetic dose for hypnosis

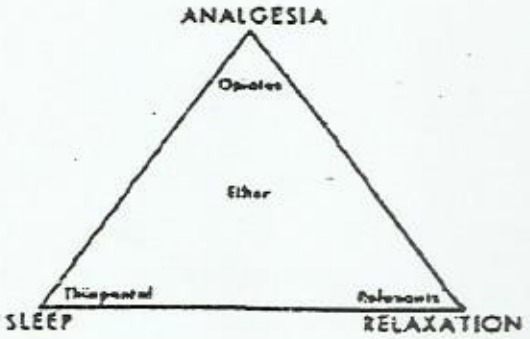

Figure 1: Balanced Anaesthesia represented as a triangle

Classification of Drugs Used for G.A.

Most of the anaesthetic drugs are very potent, thus they must be given in the exact dose that produces the desired effect to avoid toxicity by overdose or awakening of the patient by underdose. The effect of any given dose must therefore be predictable and immediate which can only be insured by inhalational or I.V routes of administration. The inhalational route, due to absorption through the huge alveolar surface, is so fast and complete, that the effect of the given concentration is as immediate as if it were given by the I.V. route. Thus the anaesthetic effect will be immediate and maximum and can be adjusted to the desired level.

On the other hand, absorption from other routes of administration as oral, subcutaneous, intramuscular, etc... will be both unpredictable and erratic, depending on many factors which are difficult to control, making these routes unsuitable for administration of G.A drugs.

Also due to the high potency of G.A drugs used nowadays, whether by inhalation or I.V, the use of a very accurate and calibrated methods of administration. Isolation becomes very essential.

G.A. drugs are classified into 2 groups: Inhalational Agents and IV Agents

(A) Inhalational Agents

Historically, these agents were the first used to produce G.A, most of which are of historical interest and not used nowadays due to the introduction of much more refined agents (though more expensive). The reasons for discontinuing the use of some of these agents include:

a. Unpleasant odour: e.g. ether, trichlorethylene and ethyl chloride. This may render induction with these agents unpleasant, prolonged and may result in retching or vomiting.
b. Explosive potential: e.g. ether, ethyl chloride and cyclopropane. The risk is higher in the presence of electric sparks from electrical appliances in the operating theatre or even from static electricity collecting over nonconductive surfaces.
c. Organ toxicity: e.g.:
    i. Liver toxicity from chloroform
    ii. Nerve toxicity from trichlorethylene with sodalime
    iii. Nephrotoxicity from methoxyflurane.

The inhalational anaesthetic agents in use are either:

a. Vapors of volatile liquids stored in glass bottles as liquids at room temperature, vaporized and delivered to the patient in the exact required concentration through very accurately calibrated vaporizers.
b. The agent may exist in the form of a gas at room temperature, stored in cylinders and given to the patient in the required conc. Mixed with oxygen through a well-calibrated anaesthetic machine e.g. nitrous oxide "NO_2".

The inhalational agents currently used are:

1. **Nitrous Oxide**

Stored compressed at 50bar in blue cylinders as liquid. Sweet smelling, non-irritant and colorless gas, not flammable or explosive. It is a good analgesic but weak anaesthetic agent, thus when used in G.A, it must be combined with other agents, since at least 30% oxygen must be added to the inspired gas mixture to avoid hypoxia.

Historically, 100% concentration of "NO_2" was used in dentistry for short surgery but the accompanying hypoxia caused by the exclusion of oxygen was very dangerous and this method is prohibited in modern practice. In balanced anaesthetic techniques, NO_2 is used together with other inhalational agents like halothane or isoflurane with analgesics and muscle relaxants. NO_2 can be used as an analgesic as e.g. in obstetrics or in conscious sedation with L.A. in dentistry, given via nasal mask in 50% conc. With oxygen. It is not metabolized in the body and excreted unchanged. Side effects were noticed during its use, e.g.:

- Diffusion hypoxia: At the end of anaesthesia, when NO_2-oxygen mixture is replaced by air, as NO_2 diffuses through the alveoli faster than nitrogen in the air is absorbed, resulting in minimal hypoxia for up to 10 minutes. This must be corrected by giving a higher conc. of oxygen.
- Effect on closed gas spaces: E.g. bowel, pleural or peritoneal cavities, volume increases as NO_2 diffuses in faster than nitrogen diffuses out. In a compact space like the sinuses or the middle ear, pressure also increases. This may also increase the size of any air embolus.
- Cardio-vascular depression: Only significant in patients with poor myocardial contractility and high sympathadrenal activity.
- Toxicity: Affects vitamin B12 synthesis and folic acid metaboism and impairs DNA synthesis, causing bone marrow aplasia and agranulocytosis, only after very prolonged exposure. Occupational exposure may cause myelonephropathy.
- Teratogenic changes: Observed only in pregnant rats after prolonged exposure, no similar effect in man.

2. Halothane

Colorless liquid with relatively pleasant smell, decomposed by light, stored in dark bottles with 0.01% thymol added for stability. Induction of anaesthesia is relatively rapid, using inspired concentration 2 to 3%, minimal anaesthetic concentration (MAC) in pulmonary alveoli that will produce surgical anaesthesia is 0.75%, 20% is metabolized in the liver and products are excreted in urine.

During induction, there is rapid loss of pharyngeal and laryngeal reflexes with inhibition of salivary and bronchial secretions. There is an increase in respiratory rate and reduction in tidal volume and it reduces bronchospasm in patients with bronchoconstriction due to ß-mimetic effect. It is a potent depressant of myocardial contractility with dose-related reduction of cardiac output, especially with controlled ventilation; the hypotensive effect is augmented by bradycardia, which can be antagonized by atropine. Arrythmias are very common due to the increased myocardial excitability augmented by hypercarbia, hypoxia or increased circulating catecholamines or due to bradycardia caused by central vagal stimulation. This becomes more serious when using L.A. with adrenaline and may end by cardiac arrest, avoided by avoiding hypoxia, hypercarbia, and adrenaline conc. More than 1:100.000 and using not more than 10ml of this in any 10 minutes.

Post-operative nausea and vomiting are rare, because it inhibits gastrointestinal motility. It relaxes the uterine muscles, resulting in postpartum hemorrhage during Caesarian section or therapeutic abortion. It causes moderate muscle relaxation, Halothane hepatitis with jaundice and liver failure that can be fatal and rarely occurs in predisposed patients, especially with repeated use of Halothane in obese patients under hypoxia and with enzyme induction by phenobarbitone or phenytoin.

3. Enflurone

Colorless liquid with ethereal odor, stable in light. Induction of anaesthesia and recovery are both rapid due to its high potency, it may cause marked C.N.S stimulation as shown by the E.E.G., potentiated by reduction in PCO2 "Carbon Dioxide Tension" and manifested by episodes of tonic or clonic twitches of the jaws, E.E.G changes may continue for up to 30 days following anaesthesia.

In the C.V.S., it may cause some hypotension, with tachycardia with reduction in cardiac output and stroke volume, which is proportional to the depth of anaesthesia and more with controlled ventilation than in spontaneous breathing. Arrythmias are rare.

Respiration is depressed more than with halothane or isoflurane. Muscle relaxation is marked, thus smaller doses of muscle relaxant drugs will be needed during surgery. On liver and kidney, cases of hepatitis do occur but less than with halothane. It reduces renal blood flow and thus better avoided with renal diseases. Anaesthesia: 3-4% conc. can be used for induction, while 1-3% for maintenance, reduced if NO_2 is used. It is mainly suitable for patients with risk of cardiac arrythmias.

4. Isoflurane

Colorless, ethereal liquid, stable in light. On C.N.S, unlike influrone, it has no convulsant effect, even with low PCO2 and it increases the cerebral blood flow. Respiration is greatly depressed with rise in PCO2 more than with halothane. This is greatly countered by surgical stimulation. Little effect on cardiac contractility, arterial bl.pr falls slightly due to reduction in peripheral resistance but cardiac output is not greatly affected. Risk of arrythmias is rare, it is a potent coronary vasodilator, therefore it may cause redistribution of myocardial blood flow with resultant ischaemia, thus better avoided in coronary heart hiseases, especially those associated with left ventricular failure.

Good muscle relaxant and also potentiates the effect of muscle relaxants. Liver is not affected, some reduction in renal blood flow that recovers fully after anaesthesia.

Anaesthesia: ideal agent with marked muscle relaxation and stable C.V.S., induction and recovery are very rapid but pungent odor may cause breath holding, delaying induction. 1.5% can be used for maintenance, reduced if NO_2 is used.

5. **Sevoflurane**

Induction and recovery are more rapid than with any other agent. C.V.S. very stable, non-irritant to the respiratory tract but is a powerful respiratory depressant. Effect on cerebral blood flow and O_2 consumption are similar. Slightly metabolized in the body. Very expensive.

(B) Intravenous Anaesthetic Agents

G.A. can be produced by many C.N.S depressant drugs e.g. sedatives, hypnotics and tranquilizers and narcotics but only if given in doses high enough to produce cardiovascular and respiratory depression and delayed recovery, only few drugs are suitable to produce G.A by I.V route and are used mainly for induction of anaesthesia which become more rapid and smoother than with inhalational agents.

They can also be used for maintenance of anaesthesia, given in repeated bolus doses or as continuous I.V infusion. Used also for sedation and treatment of epilepsy.

Properties of the Ideal IV Anaesthetic Agent

1. Rapid onset, resulting in smooth and rapid induction.
2. Rapid recovery, especially for outpatient use, e.g. in the dental clinic.
3. Produces analgesia in smaller doses.
4. Minimal or no cardiovascular or respiratory depression.
5. Not emetic.
6. No excitatory effect, e.g. coughing, hiccup or involuntary movement.
7. No emergence phenomenas, e.g. nightmares.
8. No pain on injection or venous sequelae and safe if it enters an artery by accident.
9. No toxic effects, histamine release or hypersensitivity reactions.
10. Water soluble and long shelf life.
11. No stimulation of parasympathetic.
12. Not cumulative.

Classification of IV Anaesthetics

A. Rapidly acting agents
   1. Barbiturates: methohexitone and thiopentone
   2. Imidazole compounds e.g. etomidate
   3. Alkyl phenols e.g. propofol
   4. Steroids, e.g. althesin and eugenols e.g. propanidol (not used nowadays)

B. Slower acting agents
   1. Ketamine or Ketalor
   2. Benzodiazepines, e.g. diazepam, lorazepam, midazolam
   3. Opioids, e.g. fentanyl, alfentanyl, sulfentanyl, morphine and pethidine.
   4. Neuroleptics: opioid + neuroleptic, e.g. droperidol

Barbiturates

Amylobarbitone and pentobarbitone were used but have been discontinued due to their unpredictable effect and prolonged recovery. The ones used now are:

a. **Thiopentone Sodium**

The sulphus analogue of pentobarbitone, dissolved in distilled water to produce 2.5% solution with pH 10.8. The dose in adults and children is 5-7mg/kg, in the elderly only 3mg/kg. In adults, 2ml are given first and the patient is asked about the presence of any local pain to avoid intra-arterial injection before giving the rest of the dose.

Induction is smooth, side effects are related to peak blood conc., thus if there is risk of C.V depression, it should be given very slowly. Supplementary doses may be given to prolong the time of G.A. for short surgical procedures, but if large doses are given, recovery will be markedly prolonged due to its cumulative effect.

Pharmacological Effects:

1. Progressive C.N.S depression, ending in G.A for a few minutes for the single dose.
2. Potent hypnotic but poor analgesic action.
3. Consciousness regained in 5-10 min.
4. Potent anticonvulsent, thus useful in treatment of epileptic attacks and toxicity from L.A.
5. Depresses on both sympathetic and parasympathetic systems, thus bradycardia may develop, but more often tachycardia occurs due to the modest hypotension that usually results.
6. On C.V.S.: depressed myocardial contractility and peripheral V.D. will result in hypotension that may be profound if a large dose is rapidly injected, especially in presence of added hypovolemia or cardiac disease.
7. On respiration: Causes ventilatory depression and a short period of apnea is common, usually preceded by few deep breaths.
   - Respiratory depression is more common in premeditated patients, especially with opioids and may need assisted or controlled ventilation to treat.
   - Bronchospasm is common but laryngospasm may be started by surgical stimulation or the presence of secretions or blood in an airway.
8. On skeletal muscles: Tone is depressed but muscle relaxation is poor, resulting in response to surgical stimulation
9. Uterine contractions only suppressed with large doses, it crosses the placental barrier, but foetal blood conc. is less than that of the mother.
10. On the eye: The intra-ocular pressure is reduced, while the pupils first dilate then constrict with surgical anaesthesia.
11. Hepatorenal functions: Impaired transiently.

Side Effects:

1. Hypotension: more with large doses, hypovolemic, shocked or hypertensive patients. Risk reduced by giving the drug slowly and never with patient sitting and avoid high dosage.
2. Respiratory depression, bronchospasm and laryngospasm treated by assisted or controlled ventilation and bronchodilator.
3. Local tissue necrosis: due to perivenous injection of this very alkaline solution. In the anticubital fossa, the median nerve may be damaged. If that happens, leave the needle in place and inject hyaluronidase, xylocaine for pain and hot fomentations; do not use 5% sol. and avoid using the anticubital fossa for injection.
4. Thrombophlebitis: Uncommon, but occurs more with 5% solution
5. Allergic reactions: Range from skin rash to fatal anaphylactic shock with C.V collapse. (very rare) avoided by sensitivity tests.
6. Intra-arterial injection: due to inadvertent injection in the brachial or aberrant ulnar artery in the anticubital fossa.

- Intense pain results "stop the injection at once", the forearm and hand become blanched and distal blisters may appear.
- There is profound spasm of the artery with local release of adrenaline, this together with endarteritis and platelet emboli and damaged red cells will cause thrombosis of the artery resulting in ischaemia and maybe gangrene in parts of the forearm, hands or fingers.
- Managed by leaving the needle in the artery and give a vasodilator-like papaverine 20mg and xylocaine around the artery to reduce pain and spasm.
- Stellate ganglion or brachial plexus block will also help to promote peripheral vasodilation, heparin given I.V. and oral anticoagulants given later and remove thrombus from artery if possible, prophylactically avoided by careful I.V. technique, avoid the anticubital fossa, draw blood first to ensure vein (dark blood, not under pressure arterial blood is bright red and under pressure). Give few test drops first and avoid using 5% solution of the drug.

b. **Methohexitone Sodium**
1% solution in distilled water is used with pH 11.1; dose is 1-1.5mg/kg in adults but less in elderly and neonates. Can be given 6.6mg/kg by I.M route or 20-25mg/kg rectally for sedation in children. Used mainly when rapid recovery is desired e.g. E.C.T, outpatient dental surgery and other minor procedures. Recovery occurs in 2-3 min. but patient remains drowsy for several hours.

Hypotension is less than with Thiopentone and cumulation is less likely after repeated doses. However, some excitatory muscle movements may occur during induction with coughing and hiccups but can be reduced by opioid premedication. Other local and systemic side effects of thiopentone can occur, but to a much lesser degree.

Non-barbiturate IV Anaesthetic

a. **Propofol "Deprivan"**
Expensive phenol derivative, given I.V as emulsion in 20 ml ampules containing 200mg, producing anaesthesia in 10-30 seconds. Used with caution in epileptics as it may produce convulsions. The recovery from unconsciousness is rapid with minimal hangover. Hypotension is more than with thiopentone and is mainly due to VD, it can be reduced by slowing the rate of IV injection, there is slight tachycardia. Apnea is longer and more common than with thiopenton, given by infusion, it reduces the tidal volume and increases the resp. rate, esp. after opioid premedication. Broncho- and laryngiospasm are uncommon.

It reduces muscle tone but has no effect on G.I.T motility. It produces transient decrease in renal and hepatic blood flow and reduces plasma level of cortisol. Induction dose in adults is 2-2.5mg/kg, reduced in elderly and increased in children to 3-3.5mg/kg. Dose for sedation during L.A. or endoscopy is 50-150µg/kg/hr. but increased to 250µ/kg/hr to supplement NO_2/O_2 anaesthesia for surgical procedures. Opioid premedications can greatly reduce these doses. Pain on injection occurs and can be reduced by using a large vein and 10mg lidocaine IV first slowly. Also some skin rashes may occur but severe reactions are rare. The rapid recovery makes it the drug of choice for outpatient G.A.; patient can be safely discharged after 2hrs.

b. **Etomidate**
Carboxylated Imidazole compound in 10ml ampules containing 20mg with pH 8.1. It is a rapidly acting G.A drug with short duration of only 2-3 min. Minimal C.V depressing effect, lesser resp. depression, but more lowering of cortisol level which will affect immunity after prolonged administration in the I.T.U.

Induction dose is 0.3mg/kg, given I.V into a large vein to avoid pain. Moderate to severe involuntary movements occur in 40% of cases, while cough and hiccup in 10%, reduced by opioid premedication. Pain on injection can be reduced by I.V injection of 10% lidocaine. Nausea and vomiting are frequent

(30%); also restlessness and delirium during recovery may occur. Venous thrombosis is also more common

c. **Ketamine Hydrochloride**

Phencyclidine derivative, that produces dissociative anaesthesia, rather than the generalized C.N.S depression produced by other anaesthetic agents. Available as isotonic sol. of pH 3.5-5.5, containing 10, 50 or 100mg/ml. Given I.V., it induces anaesthesia in 30-60 sec., while unconsciousness lasts 10-15min. If given I.M, it acts in 3-4min. and lasts for 15-25min. It is a potent analgesic and produces amnesia for up to 1hr after recovery.

Induction of G.A is smooth but emergence delirium may occur with restlessness, disorientation and agitation with vivid and often unpleasant nightmares or hallucinations that may last for up to 24 hrs. These reactions can be reduced by avoiding verbal and tactile contact with the patient during recovery and the use of narcotic or tranquilizer premedications. These reactions are less frequent in children or the elderly. It also increases cerebral blood flow and intra-cranial pressure.

On C.V.S., it increases the blood pressure and heart rate and cardiac output, with increased myocardial sensitivity to adrenaline. On respiration: Transient apnea followed by normal ventilation with maintained pharyngeal and laryngeal reflexes and a patent airway, but not always. Also salivary secretion is excessive and bronchial muscles dilated. Muscle tone increased and spontaneous movement may occur. It crosses the placental barrier to the foetus. It increases the intra-occular pr. and eyeball movement may persist during surgical anaesthesia.

Dose is 2mg/kg I.V slowly, additional 1-1.5mg/kg every 5-0min. The I.M. dose is 8-10mg/kg, while the analgesic dose is 0.25-0.5mg/kg or an infusion of 50µg/kg/min. without loss of consciousness. Useful drug in shocked patients and since most of these patients are heavily sedated post-operatively, the risk of nightmares is minimized. Also in pediatric surgery, given I.V or I.M., Also, management of trauma in difficult locations as anaesthesia or analgesia which can also be used for painful procedures as wound dressings. Contraindicated in:

i. Airway obstruction
ii. Hypertensive patients with ischaemic heart disease.
iii. Raised intracranial tension.

The prolonged recovery and emergence reactions make unsuitable for outpatient surgery in adults.

Hazards and complications of anesthesia

I- NEUROSUGICAL COMPLICATIONS.

1- CEREBRAL HYPOXIA:

==Hypoxic hypoxia: may result from

=Reduced o2 percentage in the inhaled gas mixture.
=Respiratory obstruction

==Diffusion hypoxia: in a case of pulmonary edema .

==Stagnant hypoxia: in peripheral circulatory failure.

==Histotoxic hypoxia: in poisoning.

The clinical picture of cerebral hypoxia:

-Mild cerebral hypoxia can cause delayed recovery.

-Sever hypoxia can give rise to a personality changes which may turn the normal person into idiot.

-Gross hypoxia can cause coma and death.

Factors affect the outcome of cerebral hypoxia:

A- Associated circulatory failure.
B-The presence of cerebral disease.

Treatment

=Ventilation (positive pressure) of the lung with 100% O2

- Dehydrating measures and steroids for treatment of oedema.
- Hypothermia to reduce the O_2 requirement.
- Correction of fluid balance.

2- Peripheral nerve palsy:

Causes:

a. Over stretching e.g. mal positioning especially of the upper arm, severe abduction may lead to brachial plexus palsy.

b. Compression of the nerve at the sharp edge of the operating table may lead to unlnar nerve palsy.

c. Injection: damage of the nerve by the tip of a needle e.g. median nerve palsy.

3. Convulsions:

May occur after local analgesia or general anesthesia predisposing factors include:

- Age: More vulnerable in children than adult,
- Diseases: Sepsis and hyperpyrexia.
- Pain
- Toxicity of local analgesics.

II- Cardiovascular complications:

1. Cardiovascular instability:

This includes: Hypertension – hypotension and cardiac dysrhthmias.

a- Acute hypertension:

The most common cause are:

1. Light plane of anesthesia and inadequate analgesia, there is associated lacrimation, sweating, tachycardia.
2. Hypercurbia (Co_2 accumulation) due to inadequate ventilation.
3. Undiagnosed pheochromocytoma.
4. Drugs: Most anesthetic drugs cause hypotension.

b- Acute hypotension:

May result from surgical or anesthetic causes:

. *Surgical causes:*

- Sudden changes in positions: antitrendlenburg, prone or lateral positions may cause hypotension.
- Major blood less especially in extremes of age.
- Surgical manipulations: e.g. surgical procedures at carotid body region, hilum of the lung, gall bladder, stretch or dilatation of urethra, anus or cervix.
- Following the release of tourniquet or clamps on a major vessel (release of metabolic products in the general circulation and its effect is vasodilatation).

. *Anesthetic cause:*

- Overdose of anethetic drugs, premedicants, intravenous inhalation anesthetics and also local analgesica.
- Late stage of hypoxia and hypercarbia.
- Excessive positive pressure ventilation of the lungs impedes venous return with the resultant low cardiac output.

C- Cardiac dysrhythmias:

Causes:

- Anesthetic agents especially halogenated anesthetics, they sensitize the myocardium to the action of circulating catecholamines.
- Hypoxia, hypercarbia and hypotension.
- Surgical manipulations.
- Cardiac patients are specially susceptible.

III- Respiratory complications:

1. Respiratory obstruction:

It can occur at any level of the respiratory tract:

A- In the pharynx:

The commonest site in non intubating patient. The jaw relaxes as consciousness is last and the longue falls back against the posterior pharyngeal wall. Correction by holding the jaw forwards and inserting an airway.

B- In the larynx:

1- Spasm: direct laryngeal spasm arises from irritation of the cords by too strong anesthetic vapour, or by saliva or vomit.

Reflex spasm can arise from a stimulus from the operative site especially under light anesthesia.

Treatment by pure oxygen and remove the cause of spasm.

In sever cases intubation of trachea may be indicated with the aid of short acting muscle relaxant.

2- Tumours: may cause complete obstruction when the cords relax under anesthesia preoperative signs of obstruction are indication for trachea story under local analgesia.

C- in the trachea and branch:

Profuse secretion, sputum, foregin bodies or vomit obstruct and the bronchial tree. These must be cleared by suction and the lungs filled with O_2. Bronchoscopy may be necessary. Bronchospam 9bronchial asthma) is another cause of respiratory obstruction and should receive immediate treatment.

D- In the alveoli:

Pulmonary oedema, as in heart failure, leads to impaired diffusion across the alveoli.

Signs of respiratory obstruction:

- With spontaneous respiration: noisy breathing, absent breath sounds, diminished chest movement, forced expiration or paradoxical chest movement.
- With controlled respiration: increse in pressure required to inflate the lungs.
- Signs due to hypoxia and hypercarbia: cyanosis, tachycardia, sweating and hypertension.

2. Respiratory depression:

This can result from:

Drugs: analgesics, overdose of anesthetics, muscle relaxants.

Diseases: as metabolic disorders (uraemia, diabetes), cerebral haemorrhage.

Extremes of temperature: hypothermia.

3. Postoperative pulmonary complications:

Tracheitis, bronchitis, bronchopneumonia, lung abscess and pulmonary collapse the predisposing factors an:

- Pre-existing lung disease.
- Site of operation: high incidence in upper abdominal operation.
- Anesthetic drugs and techniques with insufficient ventilation will increase the risk.

IV- Vomiting and regurgitation:

Stomach contents can reach the pharynx and inhaled by two quite different mechanisms.

1. By active vomiting during induction with intravenous or with inhalation agents.
2. By passive regurgitation as a result of result of relaxation occurring under muscle relaxants or deep general anesthesia.

Factors contributing to vomiting and regurgitation:

a. A high intragastric pressure, e.g. full stomach.
b. A high intraabdominal pressure, e.g. distended abdomen, ascites or tumours.

3. Obstruction of airway.
4. Intra-abdominal manipulation.
5. Hypoxia.

Both vomiting and regurgitation produce irritation and inflammation of the lungs up to sever hypoxia which lead to cardiac arrest. Latter on infective sequelae as bronchopneumonia and lung abscess can occur.

Prevention:

1. Empty stomach: Is the important preventive measure:
   a. In elective operations: the patient should be table nothing by month for at least 4 hours.
   b. In emergency situations: A large stomach tube is passed before induction to reduce the volume of stomach contents..
2. Measure during induction:
   a. Inhalation induction must be smoothly done with head down and to one site so that vomitus will flow away from the larynx. This is to be followed by intubation with a cuffed tube.
   b. Intubation under muscle relaxation may lead to passive regurgitation so tilt head-up to offer protection by gravity.
3. After operation:
   - Careful suction is essential, the tonsiller position is always adopted when there is a possibility of blood, secretion or vomitus to enter the tracheobronchial ree.

Treatment:

If vomiting occurs, immediately put the patient in the head down position, perform suction and give O_2, bronchoscopy and bronchial toilet as sometimes advisable.

Hydrocortisone to combat bronchial spasm and antibiotics to prevent the subsequent bronchpneumonic changes.

Regional Anaesthesia

Prof : Ibrahim Abbas

Subarachnoid block (SAB)

Definition: a sort of regional analgesia involving a relatively large part, produced by injection of a local analgesic into subarachnoid space i.e. it produces it's action by blocking the nerve roots in the subaracnoid space.

Drugs used:

There are tow main groups of local anaesthetics:

The amide group: Bupivacaine, Prilocaine, Ropivacaine and Lidocaine.

The ester group: Cocaine, Procaine and Tetracaine.

Technique:

Three parts A, B, and C.

An intravenous access and intravenous infusion must be instituted before lumbar puncture is performed.

A: lumbar puncture:

1-Sterlized set of spinal needle, syringe and vial containing the local anaesthetic is ready.

2-The anaesthetist is prepared as if he is going to perform a major operation.

3-The patient is either sitting or laying down on his side.

4-Wide area of his back including the sight of puncture and iliac crests is painted with iodine. The patient is asked to flex his back as much as possible by bringing his chin to his knee.

5-The highest part of iliac crest is palpated and an imaginary line passing from it to the highest part of the opposite iliac crest passes through the space between the third and fourth lumbar spines.

6-The site of puncture is usually between the second and the third, or third and fourth spines. The lower spine of

the chosen space is palpated and the needle introduced just above it and directed forwards with slight cephalic inclination.

7-As the needle is advanced, the puncture of the ligamentum flavum gives a characteristic sudden loss of resistance slogh more introduction of the needle is going to puncture the dura and arachnoid mater.

8- Withdraw the stillete, the C.S.F. will come down as crystal clear warm drops.

B- Injection of local anaesthetic:

attach the syringe containing the local analgesic and inject it.

C- The position after injection:

when isobaric solution is used, the patient thereafter lies supine in a horizontal position or slight Trendlenburg with a pillow under the head.

When hyperbaric solution is used the technique of course must be modified.

If the nerves to the perineum are to be blocked, the patient should remain sitting until the local anaesthetic, which is heavier than C.S.F., spreads downwards to block the nerve roots of the Cauda equina.

If the nerves to the abdomen are to be blocked, the patient lies on his back with the head downward, the degree of tilt of the table depends on the level of analgesia required, e.g. for upper abdominal operations the Trendlenburg position is more to allow the heavy fluid to gravitate cephalwards.

In hypobaric solution the opposite positions to that of hyperbaric are expected to be followed.

EFFECTS OF S.A.B. :

A- Nervous system:

S.A.B. produces paralysis of the nerve fibres in this order: First sympathetic fibres,
Then sensory fibres,
And lastly the motor fibres.

B- Cardiovascular system:

Fall of blood pressure and bradycardia due to paralysis of the sympathetic fibres from the thoracolumbar outflow, while the vagus nerve is left intact.

If this fall of blood pressure is marked (more than 50% of its original level) the patient is considered to be in a state of spinal shock.

Treatment of spinal shock: support circulation and respiration i.e

1- O2 inhalation
2- Trendelenburg position: to ensure adequate blood supply to central nervous system and improve venous return to the heart.
3- Vasoconstrictor drugs: To overcome the peripheral vasodilatation caused by sympathetic shock, e.g.
   Epinephrine 30 mg I.V. + 30 mg I.M.
   Noreadrenaline drip 1/250000
4- I.V. fluid: blood, plasma or plasma substitutes.

C- Respiratory system:

By ascending paralysis, the intercostal muscles which are supplied by the thorathic nerves are

paralysed. If the local anaesthetic solution extends to the cervical region, paralysis of the phrenic nerve which supply the diaphragm occurs. The patient may stop breathing so that respiratory support by IPPV and if necessary, tracheal intubation may be required.

D- Gastrointestinal tract:
Nausia and vomiting are common, perhapse because of the unopposed vagal tone or hypotention that decreases cerebral blood flow. Anticholinergic medication or blood pressure elevation may be used to treat this side effect.

E- Metabolic and hormonal:
S.A.B. attenuates metabolic and hormonal stress responses to surgery ;which are increase blood glucose ,cortizol, caticolamines, ADH, rennine levels,and postoperative negative nitrogen balance.

Advantages of spinal analgesia:

A- For the patient:
1- fulfilling the wish of the patient to be conscious.
2- Little postoperative troubles

B- For the surgeon:
-Ideal operating conditions:
a- complete muscular relaxation
b- contracted gut
c- quit respiration
d- diminished bleeding.

C- For the anaesthetist:
1- simple and relatively easy technique
2- wide margin of safty if sufficient attention is paid.

Disadvantages of spinal anaesthesia:

A- For the patient:
1- the lumbar puncture itself.
2- Postoperative headache and neurological complications.
3- Being conscious during operation , but this can be overcome by sedatives.

B- For the surgeon:
1- limited field of operation.
2- limited time.

C- For the anaesthetist:
1- lack of control, being a single dose procedure.
2- spinal shock, may be resistant to treatment.

Indications of spinal anaesthesia:

When no contraindication is present, spinal analgesia is suitable for any operation below diaphragm specially:

1- Major surgery when complete relaxation is required, diminished bleeding is preferable, much nerve plexuses are involved and operative shock is liable to occur e.g. abdominoperineal excision of rectum.
2- Rectal and transurethral surgery when atonic sphincter is required.
3- Patients with respiratory diseases.
4- Patients with diabetes and thyrotoxicosis.
5- Patients with liver or kidney diseases.

Contraindications of spinal anaesthesia:

1- Cardiovascular diseases:
   a- Hypotension: blood pressure of less than 100 mmHg.
   b- Hypertension: when associated with atherosclerosis or cardiac diseases specially in high spinal blocks.
   c- Patients with low cardiac reserve, low fixed cardiac out put ; severe stenotic valve diseases.
   d- Hypovolemia.

2- Bleeding diathesis, as hematoma may occur leading to spinal cord compression.
3- Sepsis at site of puncture.
4- Marked deformity at the back or diseases of the spine e.g. pott,s disease.
5- Mechanical factors: splenting of diaphragm by large abdominal tumour may produce anoxia if respiratory muscles have been paralyzed by high spinal block.
6- Breathless patient from any cause.
7- Gastrointestinal perforation or impending perforation, because increased pristalsis may cause soiling of the general peritoneal cavity

Complicatins of spinal anaesthesia:

1- Total spinal:
it is a local anaesthetic depression of the cervical spinal cord and brain stem includes disphonia, desnia, upper extremity weakness, loss of consciousness, pupilary dilatation, hypotension, bradicardia and cardio pulmonary arrest.
Treatment includes securing the airway, positive pressure ventilation, volume infusion and vaso pressor support.

2- Postdural puncture headache:
Severe headache may develop after dural puncture, secondary to the vent in the dura and the resultanat CSF leakage. This leads to traction on the intra cranial blood vessels and nerves.

Symptoms: characteristic headache that appears with raising the head and relieved by recumbency.

Treatment: prophylactic.

The aim is to allow but minimal leakage of CSF as possible.

a) by using the finest lumber puncture needle.
b) Patient lies in bed as long as possible with foot of bed raised.

Curative:

a) Hydration (pushing of fluid).
b) Many drugs have been used (e.g. analgesics, NISD and narcotics.

3- Local anaesthetic toxicity:
inadvertant intravascular injection of the local anaesthetics or excessive dose.
Prevention: correct technique, aspiration before injection, test dose and slow injection.

4- Hypotension:
Due to:

- Total spinal block.
- Vasovagal attack.
- Anaphylactoid reaction.
- Local anaesthetic toxicity.

5- Neurological complications:
* Neurites.
* Adhesive arachnoidites.
* Meningites and meningism.
* Heamatoma and spinal cord compression.
* Abcsess formation.

6- Urinary retention:
* Avoid over hydration.
* May need cathterisation.

7- Equipment complications:
spinal needle may break at the junction between needle and the hub.

Causes of death under spinal anaesthesia:

1- spinal shock.
2- Respiratory paralysis.
3- Usually combined.